COVID-19 Is there an end?: questions and answers

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AUSTRALIAN FEDERATION OF AIDS ORGANISATIONS
Leading the community response to HIV

AFAO, invited the following experts to present information and respond to questions

Dr Rob Grenfell is the Director of CSIRO's Health and Biosecurity Business Unit and leads a broad portfolio covering Nutrition, eHealth and Diagnostics and Biosecurity
Damien Brown is a public health medicine registrar, currently working at The Doherty Institute in Melbourne

This document provides responses to questions raised in the zoom meeting briefing delivered on April 20 2020 located here. The responses are from our speakers Dr Rob Grenfell (RG) and Damien Brown (DB). The names of people asking questions have been de-identified to protect their privacy.

Clinical Questions

Is lung damage permanent from COVID-19 infection?

It's too early to tell. In severe disease, the lungs are the site of the most damage/activity, so it'd be a fair assumption that in those who recover, there would be damage that persists for a period beyond their symptoms resolving. Whether this is permanent or not is too early to know, given that the first COVID-19 cases known were only about 4 months ago. There are many studies following up recovered patients though, including in Australia.

Are there any long-term health impacts from COVID19, or is it still too early to know?

There are case reports of heart damage, loss of smell, persisting fatigue, and some other symptoms, but again it's too early to tell – the first cases were only four months ago, so we'll know a lot more soon, especially from the huge numbers of cases in European countries and the US. Bear in mind that the overwhelming majority of cases or mild or asymptomatic, and most patients appear to recover with no issues.

What do we know about re-infection with COVID-19?

There are a few things to consider here. Firstly, although there are some reported cases of possible re-infection, this implies that the patient(s) cleared the virus and then became re-infected. We can't know for sure if they cleared the virus in the first place though. (E.g. was one of the tests incorrectly negative, giving the impression of recovery, but then correctly positive again? – this is possible. Or did the later test just pick up traces of viral genetic material (this is what it's designed to do) which was old and left over, as opposed to identifying live and active new virus?). So interpretation of these tests is an issue. Genetic typing of the viruses, to determine exactly whether they're the same between the initial and possible re-infection, will help (and is becoming available). Secondly, with other coronaviruses, immunity persists for around a year or more after infection, so hopefully this will be similar (or ideally longer). With most viruses, immunity after an infection persists for a period of time, but there are always outliers; the vast majority of people respond well to Hep B vaccinations, for example, but some never develop immunity. Immunology is complex, and we're in very early days of knowing/studying this new virus.

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Public health questions

Do we have any sense of how many people may be infected but asymptomatic? Assuming infection may lead to immunity (at least in some cases), do we have any sense of the extent to which Australia's current reduction in new infections may be due to a slowly building 'herd immunity'?

A recent study in Iceland revealed that around 1% of their population (in a randomly selected group) had evidence of prior infection. That's a pretty small number. Herd immunity would require at least 60-70% of the population to have some immunity. Australia has good control currently and we've got a good testing regime (almost half a million tests performed to date), and the number of tests that have been positive is low (less than 2% currently, and initially it was around 1:1000). So we don't seem to have a lot of transmission/spread. This is good for individuals, but bad for herd immunity – the virus almost certainly hasn't spread enough to contribute significantly to herd immunity, even if there is a reasonable pool of asymptomatic cases out there (and we'd know if there were big 'silent' pools of asymptomatic people, because some of the people they infected would become symptomatic and come to our attention).

I am wondering whether there is a measure to drive the rate of testing? Perhaps a "target" transmission rate that would not break the system. Since the asymptomatic% is anywhere between 5-80% https://www.cebm.net/covid-19/covid-19-what-proportion-are-asymptomatic (and I understand that the swabs are reserved for suspected cases, not for everyone), I am afraid whether there is "hidden" cluster that would explode and overwhelm any health systems within a month.

Hidden clusters are a risk, but there are a few things in our favour. One is that our testing is pretty good (more than 98% of tests are negative, i.e. we're looking at a lot of well people to catch the small number of cases). It's also unlikely that all people in that cluster would be asymptomatic, and the symptomatic person would likely come to the attention of healthcare providers; all of their contacts would then be traced and asked to quarantine. But you're right that we need to expand our testing, and that's now happening – the threshold for being eligible for a test has lowered a lot, and will continue to lower. 'Sentinel testing' is also likely to start soon, in which asymptomatic, otherwise well people will be tested as per a specific strategy to try and identify unknown cases. Ditto wastewater testing for viral genetic material, to pick up community spread that may not yet be known/reported.

Is COVID-19 transmissible through airborne particles?

Maybe. This is a complex area, and is being actively studied. It appears to be largely droplet transmitted (this is supported by epidemiological observations, i.e. seeing who gets infected from a positive case, and what sort of contact/proximity they had), but there may well be an airborne component to transmission. I'm not an expert in this area. But the line between droplet spread (droplets are transiently airborne, eg when coughed/sneezed etc) and airborne spread per se (smaller particles, in the air for longer), is somewhat arbitrary, and there is a degree of overlap for some pathogens. Measles is clearly airborne – you can get infected by entering a room many hours after an infected patient was there, and the R0 (number of secondary cases from that one case) is about 18. SARS-CoV-2 is about 2.5, so it's far less infectious. But with the other coronaviruses, there is an element of both, albeit tending largely towards droplet spread. This article discusses the topic in detail (open access):
https://bmcinfectdis.biomedcentral.com/articles/10.1186/s12879-019-3707-y

Would the inclusion of masks within the strategy have any impact?

As per the airborne question above, the evidence is not 100% clear, but many countries have moved towards recommending (or mandating) them. It definitely reduces the risk of an infected person (if they're wearing a mask) from spreading it to others. Whether it reduces your risk of picking it up in low transmission setting, such as Australia at the moment, is still to be confidently determined (that is, assuming you're socially distancing anyway). There is also the risk that improper handling of a mask could cause you to infect yourself too (e.g. touching your face with an infected hand while trying to apply/remove the mask). Hand washing, cough/sneeze etiquette, and physical distancing remain the mainstays of prevention and control.